expertom3ga3

# Benefits in Inflammatory Processes

Inflammation is an essential physiological process for maintaining homeostasis and tissue recovery after injury or infection. However, prolonged inflammation can be harmful and lead to tissue destruction. If after the inflammatory response, the aggression heals, normal homeostatic functions in the tissues are restored; when the acute inflammatory process is not resolved, the process becomes chronic.(1)

Novel substances have been identified, produced by enzymes from, firstly, eicosapentaenoic acid (EPA), E-series resolvins, and, secondly, docosahexaenoic acid (DHA), D-series resolvins and D1 protectin. These substances, resolvins and protectins, are compounds with actions that resolve inflammatory processes: they exhibit significant anti-inflammatory and immunoregulatory properties; they are potent endogenous anti-inflammation agonists and chemical mediators that favour resolution, lessening the inflammation and lesion mediated by the polymorphonuclear (PMN) granulocytes, involved in many of the most frequent human diseases.(2)

## Periodontitis

In periodontitis, although adequate pharmacological management of acute inflammation can prevent tissue damage, inadequate resolution and subsequent failure of the tissue to return to homeostasis produces its destruction (mediated by neutrophils) and chronic inflammation. Resolvins and protec

# Benefits in Inflammatory Processes

tins stimulate resolution of the inflammation. Resolvin E1 not only protects from inflammation-induced alveolar bone loss(3), but also induces restoration of the bone loss.(4)

## Inflammatory intestinal disease

Inflammatory response mediators (interleukin (IL) 1 and 6 and tumour necrosis factor a (TNF-a)) play a key role in the development of inflammatory intestinal disease, thus these mediators are one of the targets of pharmacological therapy.(5,6) Although current treatments for inflammatory intestinal disease are effective in reducing relapses, they can generate long-term secondary effects, in particular, a decrease in immune response.(7) The effects of long-chain Omega-3 polyunsaturated fatty acids (LC-PUFAs) in reducing concentrations of pro-inflammatory cytokines show that these molecules are important in treating intestinal inflammatory disease.(8) Studies of animals with inflammatory intestinal disease have shown that Omega-3 LC-PUFAs can substantially reduce the production of PGE2, TNF-a, LTB4 and TXA2.(9, 10)

expertom3ga3

# Benefits in Inflammatory Processes

## Bibliography


1. Nathan C. Points of control in inflammation. Nature 2002;420:846-852.
2. Serhan CN. Novel chemical mediators in the resolution of inflammation: resolvins and protectins. Anesthesiol Clin 2006;242:341-364.
3. Hasturk H, Kantarci A, Ohira T et al. RvE1 protects from local inflammation and osteoclast-mediated bone destruction in periodontitis. FASEB J 2006;202:401-418.
4. Hasturk H, Kantarci A, Goguet-Surmenian E et al. Resolvin E1 regulates inflammation at the cellular and tissue level and restores tissue homeostasis in vivo. J Immunol 2007;17910:7021-7029.
5. Bruera E, Strasser F, Palmer JL et al. Effect of fish oil on appetite and other symptoms in patients with advanced cancer and anorexia/cachexia: a double-blind placebo-controlled study. J Clin Oncol 2003;21:129-134.
6. Calder PC. Polyunsaturated fatty acids, inflammatory processes and inflammatory bowel diseases. Mol Nutr Food Res 2008;528:885-897.
7. Simopoulos AP. Omega-3 fatty acids in inflammation and autoimmune diseases. J Am Coll Nutr 2002;216:495-505.
8. Tamboli CP. Current medical therapy for chronic inflammatory bowel diseases. Surg Clin North Am 2007;873:697-725.
9. Tsekos E, Reuter C, Stehle P et al. Perioperative administration of parenteral fish oil supplements in a routine clinical setting improves patient outcome after major abdominal surgery. Clin Nutr 2004;233:325-330.
10. Kono H, Fujii H, Ogiku M et al. Enteral diets enriched with medium-chain triglycerides and N-3 fatty acids prevent chemically induced experimental colitis in rats. Transl Res 2010;156:282-291.
11. Uchiyama K, Nakamura M, Odahara S et al. N-3 polyunsaturated fatty acid diet therapy for patients with inflammatory bowel disease. Inflamm Bowel Dis 2010;16:1696-1707.
12. Parameswaran N, Patial S. Tumor necrosis factor-a signaling in macrophages. Crit Rev Eukaryot Gene Expr 2010;20:87-103.
13. McInnes IB, O'Dell JR. State of the art: rheumatoid arthritis. Ann Rheum Dis 2010;69:1898-1906.
14. Scott DL, Wolfe F, Huizinga TW. Rheumatoid arthritis. Lancet 2010;25:1094-1108.
15. Caughey GE, James MJ, Proudman SM et al. Fish oil supplementation increases the cyclooxygenase inhibitory activity of paracetamol in rheumatoid arthritis patients. Complement Ther Med 2010;18:171-174.
16. Ierna M, Kerr A, Scales H et al. Supplementation of diet with krill oil protects against experimental rheumatoid arthritis. BMC Musculoskelet Disord 2010;11:136.
17. Bahadori B, Uitz E, Thonhofer R et al. Omega-3 Fatty acids infusions as adjuvant therapy in rheumatoid arthritis. J Parenter Enteral Nutr 2010;34:151-155.
18. Hurst S, Zainal Z, Caterson B et al. Dietary fatty acids and arthritis. Prostaglandins Leukot Essent Fatty Acids 2010;82:315-318.
19. Cleland LG, French J, Betts HW et al. Clinical and biochemical effects of dietary fish oil supplements in rheumatoid arthritis. J Rheumatol 1988;15:1471-1475.
20. Dawczynski C, Schubert R, Hein G et al. Longterm moderate intervention with n-3 long-chain PUFA-supplemented dairy products: effects on pathophysiological biomarkers in patients with rheumatoid arthritis. Br J Nutr 2009;101:1517-1526.
21. Galarraga B, Ho M, Youssef HM et al. Cod liver oil n-3 fatty acids as an non-steroidal anti-inflammatory drug sparing agent in rheumatoid arthritis. Rheumatology Oxford 2008;47:665-669.